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Partial sleep deprivation does not alter processes involved in semantic word priming: Event-related potential evidence

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ABSTRACT

Sleep deprivation has generally been observed to have a detrimental effect on tasks that require sustained attention for successful performance. It might however be possible to counter these effects by altering cognitive strategies. A recent semantic word priming study indicated that subjects used an effortful predictive-expectancy search of semantic memory following normal sleep, but changed to an automatic, effortless strategy following total sleep deprivation. Partial sleep deprivation occurs much more frequently than total sleep deprivation. The present study therefore employed a similar priming task following either 4 h of sleep or following normal sleep. The purpose of the study was to determine whether partial sleep deprivation would also lead to a shift in cognitive strategy to compensate for an inability to sustain attention and effortful processing necessary for using the predicative expectancy strategy. Sixteen subjects were presented with word pairs, a prime and a target that were either strongly semantically associated (*cat. . .dog*), weakly associated (*cow. . .barn*) or not associated (*apple. . .road*). The subject's task was to determine if the target word was semantically associated to the prime. A strong priming effect was observed in both conditions. RTs were slower, accuracy lower, and N400 larger to unassociated targets, independent of the amount of sleep. The overall N400 did not differ as a function of sleep. The scalp distribution of the N400 was also similar following both normal sleep and sleep loss. There was thus little evidence of a difference in the processing of the target stimulus as a function of the amount sleep. Similarly, ERPs in the period between the onset of the prime and the subsequent target also did not differ between the normal sleep and sleep loss conditions. In contrast to total sleep deprivation, subjects therefore appeared to use a common predictive expectancy strategy in both conditions. This strategy does however require an effortful sustaining of attention, and may not have been entirely successful when sleep was restricted. A slight but significant decrease in accuracy was noted.

1. Introduction

Sleep deprivation has been consistently shown to degrade performance on many cognitive tasks especially for those that require sustained attention for successful completion (reviewed by Lim & Dinges, 2010). Not all studies however find this decline in performance perhaps because subjects may be able to compensate for the effects of sleep deprivation by altering cognitive strategies (Drummond et al., 2005; Lim & Dinges, 2008).

Recently, López-Zunini, Muller-Gass, and Campbell (2014) provided empirical evidence of dynamic changes in cognitive strategy after subjects had been totally sleep deprived. They employed a word priming task in which an initial word (the "prime") appeared and subsequently followed by a "target" word that was either strongly (e.g., *cat. . .dog*), weakly (*cow. . .barn*), or not semantically associated (*apple. . .road*) with the prime. The subject's task was to determine if the target was in fact semantically associated with the prime. Following normal sleep, the usual priming advantage was found. Reaction times (RTs) were faster and accuracy was higher following presentation of strongly primed targets. The strong priming advantage was still maintained following total sleep deprivation (TSD), although there was a small non-significant overall deterioration in accuracy.

Priming tasks are especially useful for the study of sleep deprivation because the similarity in performance that López-Zunini et al. observed can be obtained through the use of two very different cognitive strategies. Their results suggest that subjects might have used one strategy following normal sleep but then switch

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to a different strategy to maintain performance following total sleep deprivation. Most cognitive researchers agree that the presentation of the prime activates a semantic memory network with strongly associated semantic concepts represented in neighboring nodes and weakly associated concepts represented in more distant nodes. Targets that are not associated with the prime are not represented in the semantic network and are thus not available to memory prior to their onset (Collins & Loftus, 1975; Neely, 1991). The search is completed more rapidly for strongly associated targets than for either weakly associated or unassociated targets. The extent to which effortful processing is required to search the semantic network is disputed. One theory proposes that the subject can use the prime to intentionally predict a set of semantically associated target candidates (Becker, 1980; Neely, 1976; Posner & Snyder, 1975). There is however a cost to the use of this strategy as it requires active and effortful processing in order to generate possible target candidates and maintain them in working memory. This strategy would be appropriate following normal sleep, when sufficient cognitive resources are readily available. The use of an effortful expectancy-predictive strategy was promoted in the López-Zunini et al. study by having subjects intentionally determine if the target was associated with the prime. Moreover, the time between the onset of the prime and the target was 700 ms, long enough to permit the use of this strategy. A second theory proposes that the search of semantic memory can be initiated automatically and effortlessly, without the need for active and sustained attention (Collins & Loftus, 1975). This strategy could thus have been used to maintain performance following sleep deprivation when considerably fewer cognitive resources were presumably available.

López-Zunini et al. also recorded event-related potentials (ERPs) following the presentation of the primes and targets to monitor in real-time whether performance following sleep deprivation was maintained through the use of a similar or different cognitive strategy compared to that employed following normal sleep. In the normal sleep condition, a negative-going waveform peaking at about 400 ms (thus the "N400") following presentation of the target was much larger when it was not semantically associated with the prime, a finding replicating many others (reviewed by Kutas & Federmeier, 2011). This was also however the case in the TSD condition. Importantly, the overall N400 across all stimulus types was significantly reduced following TSD. Holcomb (1988) had previously noted that the use of an automatic, effortless search of semantic memory will result in a reduction of the N400 compared to when an effortful, intentional search is employed. Thus, the difference in the overall N400 amplitude following TSD might be explained by a change from an effortful to a relatively effortless cognitive strategy. The use of different cognitive strategies should have especially been apparent between the presentation of the prime and the subsequent target words, the time when the semantic network is searched. A long-lasting negativity beginning at about 300 ms following the prime and continuing until the onset of the target word was indeed significantly larger in the normal sleep condition. This might reflect additional effortful processing or may be a reflection of a so-called contingent negative variation (CNV) which has long been known to be attenuated following sleep deprivation (Naitoh, Johnson, & Lubin, 1971).

While total sleep deprivation conditions are often employed in the study of the cognitive effects of sleep loss, in real-life situations, total sleep deprivation occurs much less frequently than partial sleep deprivation. The American Centers for Disease Control (CDC) analyzed data obtained from interviews on more than 15,000 workers and indicated that more than 30% of its sample slept on average for less than 6 h a day (CDC, 2012). Sleep restriction is thus a hallmark of modern society due to a broad range of societal and medical factors such as recreational opportunities, work schedules and sleeping disorders. In many cases, sleep is delayed, such that the onset of sleep begins later than normal.

While partial sleep deprivation occurs much more often than total sleep deprivation, relatively few studies have examined its effects on cognitive processing. The results of studies investigating partial sleep deprivation are less consistent although tasks that require sustained attention are more likely to show a deterioration in performance (see Dinges, Rogers, & Baynard, 2005; Shekleton, Rogers, & Rajaratnam, 2010 for reviews). When partial sleep deprivation does affect performance, the effects are generally reduced compared to TSD (Casement, Broussard, Mullington, & Press, 2006; Otmani, Pebayle, Roge, & Muzet, 2005; Van Dongen, Maislin, Mullington, & Dinges, 2003). The effects may vary with the extent of sleep deprivation (Belenky, Wesensten, Thorne, et al., 2003) and can affect ERPs related to both automatic and controlled aspects of attention (Zerouali, Jemel, & Godbout, 2010).

The present study examines the effect of 4 h of sleep on the word priming task used by López-Zunini et al. The interest is whether a common strategy will be used in the normal and partial sleep condition or whether, as was the case with TSD, a different cognitive strategy will be adopted to maintain consistent performance following partial sleep deprivation.

2. Methods

2.1. Subjects

Sixteen young adults (10 females) between the ages of 20 and 30 years (Mean = 23.1, SD = 2.9 years) volunteered to participate in this study. All were right-handed, with good self-reported health, normal or corrected-to-normal eyesight and were not taking any medications known to affect cognitive function. None had any history of neurological or psychiatric disorder. Absence of sleep disorders was verified using the Pittsburgh Sleep Index (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Subjects were required to maintain a regular sleep schedule (no night or shift work). Subjects were asked to abstain from alcohol and caffeine in the 24 h period prior to the start of data collection. This study was conducted following the guidelines of the Canadian Tri-Council (Health, Natural, and Social Sciences) on ethical conduct involving humans. All subjects gave written informed consent prior to the beginning of the experiment and were paid an honorarium for their participation.

2.2. Procedure

All subjects participated in two experimental sessions, one following a normal night of sleep, and one following partial sleep deprivation. The order of the sessions was counterbalanced across subjects and at least one week separated the two sleep conditions.

Subjects were asked to retire for sleep at their normal bedtime and to awaken at their normal wake time for four consecutive nights prior to both normal sleep and sleep deprivation sessions. They completed sleep logs each morning upon awakening. The sleep logs were subsequently verified to assure compliance. The sleep patterns did not differ prior to the normal sleep and sleep deprivation sessions. On the sleep-deprived night, subjects were instructed to sleep at 03:00 and were awakened at 07:00. Subjects were required to wear a wrist actigraph on the night prior to testing. Subsequent analyses of the actigraph data did indicate that subjects followed this sleep schedule. Data collection began the following morning between 08:00 and 10:00. While awake at night, subjects played computer games or watched videos/television. Upon coming to the lab in the morning, subjects completed the Stanford Sleepiness Scale, a 7-point rating scale (1 = awake and alert, 7 = falling asleep).

2.2.1. Experimental task

Subjects engaged in a word association priming task in both testing sessions. Details of this task can be found in López-Zunini et al. (2014). In brief, two versions of the task were available, each with a different sequence of words in order to avoid stimulus repetition priming effects. Each trial started with a fixation point ("+") presented in the center of the monitor for 2000 ms. The prime word was then presented for 200 ms followed by a 500 ms interstimulus interval (ISI). A target word was then presented for a maximum of 1200 ms or until the subject responded. Three different target types were employed: strongly associated (e.g. *dog-cat*), weakly associated (e.g. *cow-barn*), and unassociated (e.g. *apple-road*) with the prime. Subjects were asked to press one mouse button if the target was semantically associated with the prime and a different button if the target was not associated with the prime. They were instructed to respond while the stimulus was still displayed (i.e., within 1200 ms). The strength of association of the prime-target pair was determined by a normative data set, the Edinburgh Associative Thesaurus (Kiss, Armstrong, Milroy, & Piper, 1973). There was no semantic association for the unassociated prime and target words.

A total of 420 prime-target pairs were constructed and were randomly divided into two lists (i.e., 210 prime-target pairs) that included 70 pairs of each of the prime-target association types. Subjects were presented with both lists, one during each experimental session. Subjects did not therefore repeatedly see the same prime-target. Half the subjects were presented with one list initially and the other half, with the other list. Stimulus presentation and response monitoring were controlled by E-prime software (Psychology Software Tools Inc., Sharpsburg, Pennsylvania) using a PC with a Windows XP operating system.

2.2.2. ERP recording

EEG activity was recorded from 32 sites using active silver–silver chloride electrodes attached to an electrode cap (Brain Products, GmbH, Munich, Germany). An EOG electrode was placed on the infra-orbital ridge of the left eye to record vertical eye movements. The nose was used as a reference for all channels.

Inter-electrode impedance varied from 20 to 50 kΩ. A high filter was set at 250 Hz. The time constant was 2 s. The physiological data were sampled every 2 ms (i.e., a 500 Hz sampling rate). Offline, the data were reconstructed using Brain Products' Analyzer2 software. Vertical EOG activity was computed by subtracting activity recorded at the FP1 channel from that recorded at the infra-orbital ridge of the eye. Horizontal EOG activity was computed by subtracting activity recorded at the FT9 channel from that at FT10. The continuous data were digitally filtered using a high filter set at 20 Hz and a low filter set at 0.08 Hz (i.e., a time constant of 2 s). The EEG was visually inspected for channels containing high levels of noise. These channels were replaced by interpolating the data of the surrounding electrode sites (Perrin, Pernier, Bertrand, & Echallier, 1989). None of the subjects had more than 4 EEG channels that required interpolation. Independent Components Analysis (Makeig, Bell, Jung, & Sejnowski, 1996) was used to identify eye movement and blinks that were statistically independent of the EEG activity. This factor was then partialled out of the EEG traces.

2.3. Data analyses

2.3.1. Behavioral performance data

Performance was measured in terms of accuracy (hit rate) and reaction time (RT). RTs longer than 1200 ms were removed from the analysis. Two-way ANOVAs with repeated measures on target association (strongly associated, weakly associated, unassociated) and sleep condition (normal sleep, partial sleep deprivation) were conducted for the accuracy and RT data.

2.3.2. ERP data

The continuous EEG activity was segmented into separate epochs for the prime and target words. Each epoch began 100 ms before the onset of the word. The prime and target epoch continued for 800 and 1200 ms respectively following stimulus onset. The single trial epochs were sorted and averaged according to electrode position and prime-target association strength. The 100 ms pre-stimulus period served as a baseline.[1] Epochs were baseline corrected prior to averaging. Only correct trials were averaged. Any epochs containing EEG activity exceeding ±100 μV on any channel were rejected from averaging. This was relatively rare because the major source of artifact, eye movements and blinks, had already been corrected in the EEG channels.

ERP components were measured relative to the average of all data points within the pre-stimulus baseline. The prime word waveform consisted of a central maximum positive deflection at about 200 ms followed by a long-lasting negativity beginning at about 300 ms. The P200 was quantified initially at Cz by averaging all data points within ±30 ms of the peak identified in the grand average and then measured using the same procedure at all other electrode sites. The long-lasting negativity was quantified as the average of all data points between 350 and 450 ms.

A P200 was again visible following presentation of the target and was quantified in the same way as the P200 elicited by the prime. A large central maximum negativity N400 was elicited by weakly associated and unassociated targets. It was also quantified initially at Cz as the average of all data points ±30 ms of the peak identified in the grand average.[2] A late positivity was also apparent after the N400. This late positivity was quantified as the average of all data points between 600 and 800 ms.

Electrode sites were grouped into regions of interest (ROIs), to include 9 electrode sites where the ERP components of interest were largest. The ROIs allowed for an analysis of an anterior–posterior and an inter-hemisphere factor. Specifically for the anterior–posterior electrode factor, three electrodes for frontal (F3, Fz, F4), central (C3, Cz, C4), and parietal (P3, Pz, P4) sites were chosen for analysis. The P200 and N400 components were thus quantified at each of these sites within the latency range identified at Cz. For the inter-hemisphere factor, three electrodes for left (F3, C3, P3), midline (Fz, Cz, Pz), and right (F4, C4, P4) sites were chosen for analysis. A four-way ANOVA with repeated measures on target association (strong, weak, unassociated), sleep (normal sleep, partial sleep deprivation), anterior–posterior (frontal, central, parietal), and hemisphere (left, midline, right) were run on each of the intervals for the prime and target data. For all statistical analyses, a Geisser-Greenhouse correction procedure was used when appropriate (Geisser & Greenhouse, 1958).

3. Results

3.1. Stanford sleepiness scale

Subjective ratings of sleepiness significantly increased following the sleep-deprived compared to the normal sleep condition. The mean subjective rating was 2.00 (SD = 0.63) following normal sleep and 3.18 (SD = 1.10) following sleep deprivation, $t(15) = 4.28$, $p < 0.001$.

[1] López-Zunini et al. employed a 125 ms post-stimulus baseline because the pre-stimulus interval was not stable and varied between the two sleep conditions. This was not the case in the present study.

[2] López-Zunini et al. measured N400 over a 300–500 ms interval because it was often small and a distinct peak difficult to discern following TSD. A distinct N400 was apparent in grand averages in the present study following partial sleep deprivation.

3.2. Behavioral data

The mean accuracy and RT data are presented in Table 1. The usual priming effect was found following both normal sleep and partial sleep deprivation. Accuracy was thus significantly affected by the target type, $F(2,30) = 22.28$, $p < 0.001$. Accuracy was higher following presentation of the strongly associated and lower following presentation of the weakly associated and unassociated targets. For all target types, accuracy did decrease following partial sleep deprivation, $F(1,15) = 6.52$, $p < 0.02$. There was no significant interaction between target type and sleep condition ($F < 1$).

Table 1
Mean (SD in parentheses) accuracy and reaction time (in ms).

Prime-target association	Accuracy		Reaction time	
	NS[a]	PSD[b]	NS	PSD
Strong	0.92 (.06)	0.89 (.08)	596 (72)	582 (72)
Weak	0.77 (.08)	0.76 (.09)	668 (74)	638 (58)
Unassociated	0.88 (.08)	0.84 (.11)	679 (70)	669 (58)

[a] Normal sleep.
[b] Partial sleep deprivation.

Likewise, the RT data revealed a similar priming effect, regardless of the amount of sleep. Thus, RTs were faster for strongly associated than for weakly associated targets and slowest for unassociated targets, $F(2,30) = 27.29$, $p < 0.001$. Surprisingly, RTs were about 17 ms faster following sleep deprivation but the difference was not significant $F(1,15) = 2.58$, $p < 0.12$. The interaction between target type and sleep condition was also not significant ($F < 1$).

3.3. Physiological data

3.3.1. Target words

The left portion of Fig. 1 illustrates ERPs to target words following normal sleep and sleep deprivation. As can be observed, target

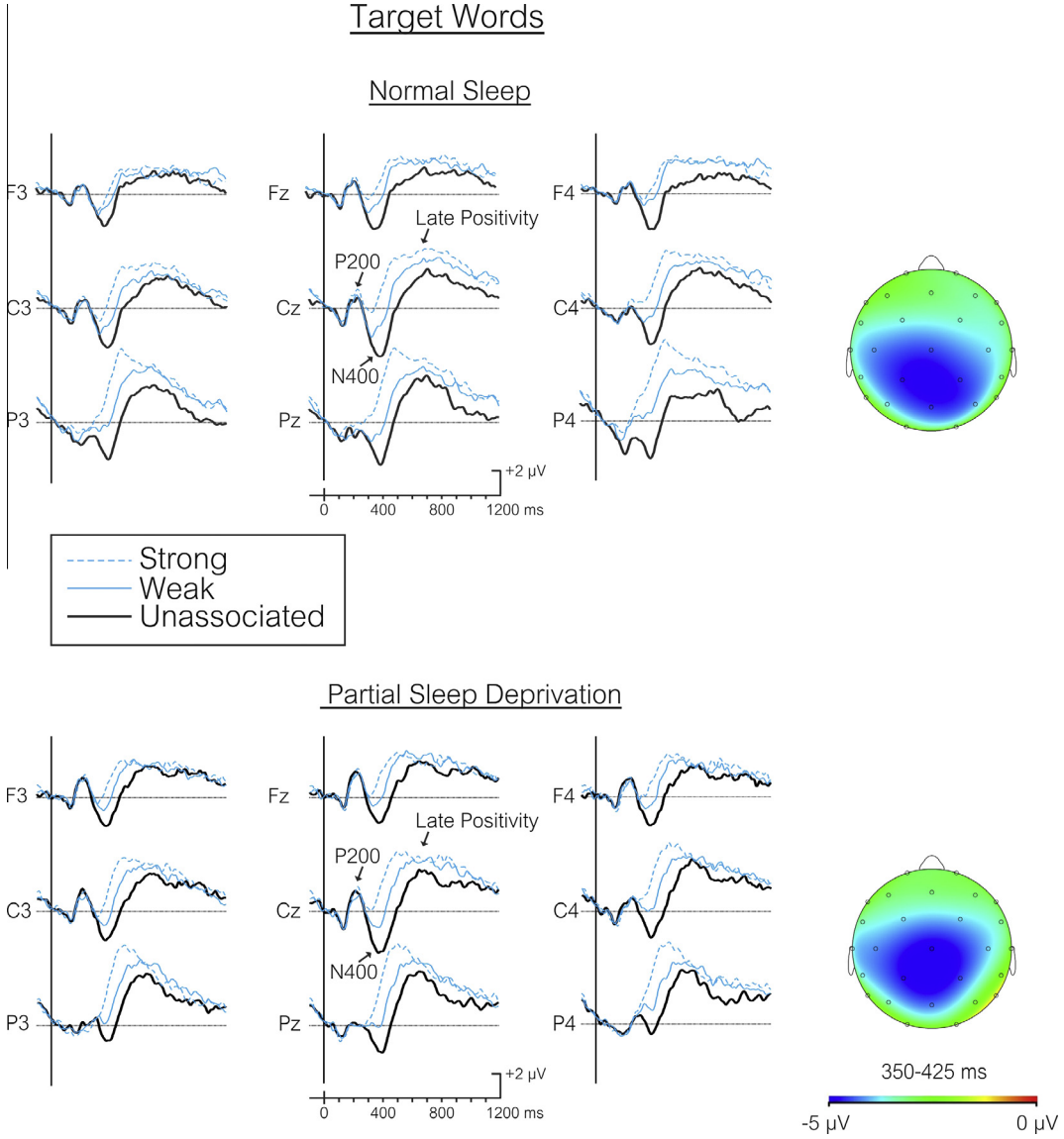

Fig. 1. Grand average ERPs to target words that were either strongly, weakly, or not associated with the prime. In this and Fig. 2, negativity at the scalp relative to the reference is indicated by a downward deflection. The early P200 was not affected by the amount of sleep. The amplitude of N400 showed a strong priming effect but was not affected by the amount of sleep. The right portion of the figure provides the spline maps of the N400 to unassociated targets. The N400 was maximum over central areas of the scalp and declined over anterior and later regions. Importantly, the scalp distribution of the N400 did not vary as a function of the amount of sleep.

Table 2
Mean (SD in parentheses) ERP amplitudes at midline electrodes following presentation of the target word.

Prime-target association	Electrodes	Condition	ERP component amplitude (in μV)		
			P200	N400	Late positivity
Strong	Fz	NS[a]	1.20 (3.23)	−0.93 (3.44)	3.51 (3.07)
		PSD[b]	2.79 (2.44)	−0.63 (1.67)	2.99 (3.16)
	Cz	NS	0.51 (3.44)	−1.68 (3.97)	5.25 (3.61)
		PSD	2.61 (2.94)	−1.10 (2.35)	4.49 (3.75)
	Pz	NS	−2.00 (4.02)	−1.04 (5.23)	5.15 (3.41)
		PSD	0.57 (3.62)	−0.86 (4.01)	4.74 (3.69)
Weak	Fz	NS	0.68 (3.60)	−2.33 (3.73)	3.13 (3.64)
		PSD	2.57 (2.68)	0.95 (3.09)	3.95 (3.62)
	Cz	NS	1.53 (3.04)	−4.36 (3.82)	3.84 (3.65)
		PSD	2.13 (3.64)	−2.58 (3.43)	4.61 (3.63)
	Pz	NS	−0.24 (3.54)	−3.41 (4.66)	4.42 (3.51)
		PSD	0.41 (3.94)	−1.70 (3.31)	5.20 (3.32)
Unassociated	Fz	NS	1.25 (3.53)	−3.60 (3.20)	2.00 (3.13)
		PSD	2.77 (2.29)	−2.89 (2.19)	3.02 (2.49)
	Cz	NS	0.34 (3.31)	−5.82 (3.34)	2.62 (3.66)
		PSD	1.41 (3.19)	−5.15 (2.81)	3.11 (2.42)
	Pz	NS	−2.07 (3.59)	−5.35 (4.02)	3.56 (3.60)
		PSD	−1.18 (3.84)	−4.31 (3.80)	3.78 (3.16)

[a] Normal sleep.
[b] Partial sleep deprivation.

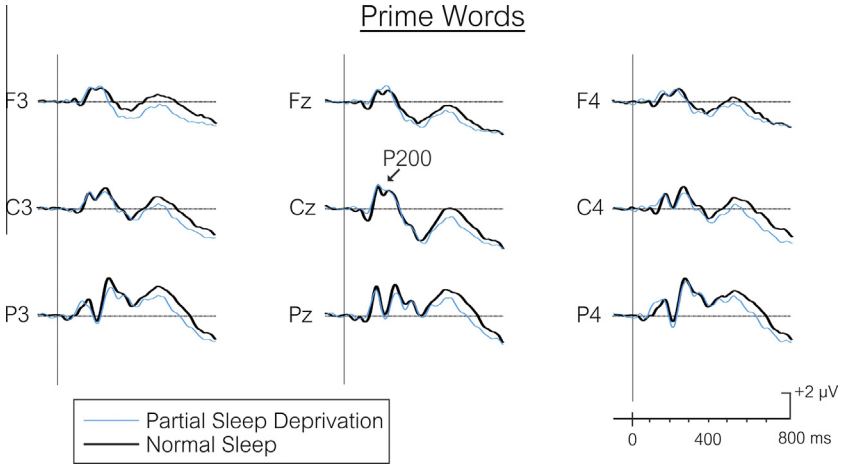

Fig. 2. Grand average ERPs to the initial prime word. Neither the P200 nor the negativity beginning at about 300 ms was affected by the amount of sleep.

ERPs consisted of an initial positivity peaking at about 200 ms (P200), followed by a large negativity at about 400 ms (N400). A long-lasting later positivity at about 600–800 ms was also apparent. The mean amplitudes of these ERP components at midline electrode sites are presented in Table 2.

The positivity in the 170–230 ms interval was largest in amplitude at fronto-central sites, declining at more posterior sites. The amplitude of the P200 did not significantly vary as a function of either target type, $F < 1$ or sleep condition, $F(1,15) = 1.87$, $p < 0.19$. The interaction between target type and sleep condition was also not significant, $F < 1$.

A main effect of target type was found, $F(2,30) = 32.96$, $p < 0.001$ on the amplitude of the N400. It was largest following presentation of the unassociated targets and smallest following presentation of the strongly associated targets. Although the overall N400 following normal sleep tended to be slightly larger than following sleep deprivation, this difference was not significant, $F(1,15) = 1.35$, $p < 0.26$. The sleep condition x target type interaction was not significant, $F < 1$.[3]

The right portion of Fig. 1 presents spline scalp distribution maps of the N400 following normal sleep and sleep deprivation. The ROI analyses did reveal significant variance in its amplitude across the scalp; N400 was largest over central regions declining in amplitude over anterior and posterior regions, $F(2,30) = 2.54$,

[3] The N400 data were also reanalyzed using a 0–125 ms post-stimulus baseline to be consistent with the scoring method used by López-Zunini et al. (2014). The results were very similar; a main effect of target type was found but the main effect of sleep deprivation and the target type X sleep interaction were not significant. The N400 data were also reanalyzed averaging all data points within a wider 300–500 ms time interval. Again, there was a main effect of target type, but no significant main effect of sleep condition. The target type X sleep interaction was also not significant. Finally, it is possible that the inclusion of the multiple electrode data factor in the ANOVA might have smeared specific N400 effects. The ANOVAs were therefore re-run only on the Cz data, where N400 was largest. The results were essentially identical.

$p < 0.09$. As is apparent in the maps of the N400, its scalp distribution did not differ between normal sleep and partial sleep loss. The ROI analyses did not reveal significant interactions between sleep condition and electrode position, $F < 1$.

For the positive waveform between 600 and 800 ms, the main effect of target type just failed to reach significance, $F(2,30) = 3.01$, $p < 0.06$; it was smallest following presentation of the unassociated compared to the weakly or strongly associated targets. Again, the amount of sleep did not however have a significant effect and the target type × sleep interaction was not significant, $F < 1$ in both cases.

3.3.2. Prime words

Fig. 2 illustrates the ERPs to the prime words following normal sleep and partial sleep deprivation. P200 was not affected by sleep condition, $F < 1$. Similarly, the long-lasting negativity following the P200 was also not affected by the amount of sleep, $F < 1$. As expected, the strength of the association of the subsequent target did not significantly affect the amplitude of either the P200 or the later negativity, $F < 1$.

4. Discussion

The purpose of this study was to investigate whether partial sleep deprivation (4 h of night sleep) would have similar effects on a priming task when compared to those found following a night of total sleep deprivation. A large priming effect was found following both normal and partial sleep. Accuracy was lower, RTs were faster and N400 larger to target words that were not semantically associated with the prime, regardless of the amount of sleep. This finding is thus consistent with those reported by López-Zunini et al. (2014). What was different in the present study was that the overall N400 was not significantly reduced in the sleep restriction condition. Similarly the scalp distribution of the N400 did not vary between the two sleep conditions. Moreover, processing between the time of the onset of the prime and the subsequent target also did not differ between the two sleep conditions. Neither the amplitude of the P200 nor the negativity between 350 and 450 ms were affected by sleep condition. Therefore, in contrast to total sleep deprivation, neither the target nor the prime ERPs revealed a difference in cognitive processing following partial sleep deprivation.

The priming paradigm was specifically designed to allow for the use of a predictive-expectancy strategy. The subject's task was to explicitly determine if the target was semantically associated with the prime. This encouraged the subject to predict possible target candidates following the presentation of the prime. To further ensure the use of this strategy, the time between the onset of the prime and the subsequent target was long (700 ms), sufficient to intentionally predict possible target candidates. The ERP evidence suggests that subjects used the predictive expectancy strategy following both normal sleep and sleep restriction. The predictive expectancy strategy does however require active and sustained attention in order to search possible target exemplars and maintain them in working memory. It would therefore appear that sufficient cognitive resources were available following partial sleep deprivation to use such an effortful, attention-demanding strategy. On the other hand, the López-Zunini et al. (2014) findings indicate that insufficient resources were available to allow for the use of this effortful strategy following TSD. Thus, in the latter case, subjects may switch to an alternate strategy, one that searches the semantic network automatically and effortlessly.

While subjects might have attempted to use the predictive-expectancy strategy following sleep restriction, the performance data indicate that it may not have been entirely successful. A slight but statistically significant deterioration (about 2.5%) in accuracy of performance was observed following sleep restriction. López-Zunini et al. found a somewhat larger decrease in accuracy (5%) but in their case, the difference failed to attain significance, perhaps due to a smaller sample size or because of a larger inter-subject variability. An alternate explanation for the present performance results is that the small loss of accuracy might have been a result of a speed-accuracy trade-off (Dorrian, Rogers, & Dinges, 2005; Gosselin, De Koninck, & Campbell, 2005; Kim, Lee, Kim, et al., 2001). RTs were slightly, but not significantly, *faster* following sleep loss. Cognitive psychologists have long noted that the accuracy and RT do interact. Accuracy of performance can be sacrificed for speed of responding. Even small changes in RT can result in large changes in accuracy (Meyer, Irwin, Osman, & Kounios, 1988). The faster RTs following partial sleep deprivation might be a result of a willingness to risk error in the accuracy of detection.

In summary, a strong priming effect was observed following normal sleep and partial sleep deprivation. The ERP measures of cognitive processing did not however differ between the two sleep conditions. In both conditions, a common effortful, predictive-expectancy strategy may thus have been adopted. This suggests that sufficient resources are available following 4 h of night sleep to maintain such effortful processing. However, this may not have been entirely successful as accuracy of responding did decrease following sleep restriction.

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